fixation-point for any pathogenic organisms which, at the moment, are circulating in the blood-stream.

It seems very probable, when all things are considered, that in any particular case more than one of the factors indicated in this communication may be operative to account for any fever which develops. Only in a comparatively few cases is it possible for one to put one's finger accurately upon the actual mechanism.

SUBCUTANEOUS EMPHYSEMA: A COMPLICATION OF INFLUENZA PNEUMONIA. REPORT OF SEVEN CASES.

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Recent clinical reports have mentioned subcutaneous emphysema as a rare complication of influenzal bronchopneumonia. Thus, Symmers[1] records 3 cases; Berkley and Coffen[2] 11; Clark and Synnott[3] 12; Blanton and Irons[4] and Stone and Swift[5] merely mention its occurrence. Leichtenstern,[6] in his exhaustive monograph on influenza, makes no note of this complication. Our own experience comprises a total of 9 cases, of which we have studied 7 cases carefully, and these are herewith presented. In about 3000 cases of influenzal pneumonia subcutaneous emphysema occurred in 0.3 per cent., and in the entire series of over 12,000 cases of influenza in only 0.07 per cent.

Clinical Features of Influenza Pneumonia. The clinical picture of influenza pneumonia has been so often described that we do not deem it necessary to repeat it here. Our experiences, on the whole, correspond to all previous reports. We desire to emphasize, however, certain features of the disease, not only because they were apparently characteristic of the picture but because they may be significant factors in the production of emphysema.

1. *Toxemia.* This was striking early in the epidemic and entirely out of proportion to all physical findings.

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[1] Jour. Am. Med. Assn., 1918, lxxi, 1482.
[2] Ibid., 1919, lxxii, 535.
[3] Am. Jour. Med. Sc., 1919, clvii, 219.
[4] Jour. Am. Med. Assn., 1918, lxxi, 1988.
[5] Ibid., 1919, lxxii, 487.
[6] Influenza, 1912. Alfred Hölder, Wien u. Leipzig.

2. *The Paroxysmal Cough.* This occurred early, was particularly distressing and closely resembled the cough of pertussis, excepting the absence of the "whoop" at the end of the paroxysms.
3. *Epistaxis and General Tendency to Hemorrhage.*
4. *The Frequency of Pain in the Chest.* The rapidity with which this developed was particularly noticeable, physical examination failing to reveal any cause.
5. *Cyanosis and Dyspnea.* The purple hue of the face, upper chest, and finger nails, at once suggested cardiac failure; but on physical examination, heart tones were generally good and there was usually no evidence of right heart failure. Blood-pressures were practically normal.
6. *The Relative Paucity of Physical Findings and the Rapidity with which the Signs of Consolidation Developed.* It is noteworthy that on admission a few crepitant rales were heard posteriorly. Percussion revealed a distinctly hyperresonant note, and this had no apparent relation to the amount of consolidation.

REPORT OF CASES.

CASE I.—Lieut. P. Taken ill and admitted October 2, 1919, with headache, sore-throat and general aching. Physical examination revealed a young, fairly well-nourished man, face congested, pulse of fair quality, temperature 103°, respirations 24. Many crepitant rales in right lower lobe; impaired resonance and diminished breath sounds.

October 4. Dulness over the right lower lobe; bronchovesicular breathing. The roentgen-ray report showed spot shadows in the lower right.

October 9. Slight cyanosis of nose, lips and ears and the physical signs of consolidation in right lower lobe. On the left side many crepitant rales. At 2 P.M. examination of the chest revealed loud crackling sounds over the apices. On palpation of these areas a fine crepitation was obtained and a diagnosis of subcutaneous emphysema made. The extent of the involvement at this time was the left supraclavicular space and upper part of the left chest. The patient had no complaint of precordial or pleuritic pain at this time. On repeated questioning he could not associate his swelling in the neck with any cough; the patient was unaware of it until it was mentioned to him by the physician. The temperature showed no alteration from that on the previous day. The respirations were not changed in rate or excursion. The roentgen-ray report on this date revealed a shadow in the right lower lobe; no evidence of pneumothorax.

October 10. The air extended bilaterally and involved the chest, neck and upper part of the abdominal wall, interfering with examination of the chest.

October 13. The emphysema now involved the left side of the face. The left eye was closed because of emphysema of the lids.

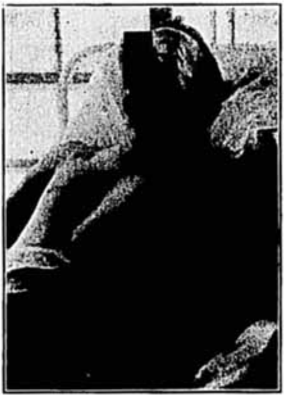

FIG. 1.—Note emphysema of left eyelid.

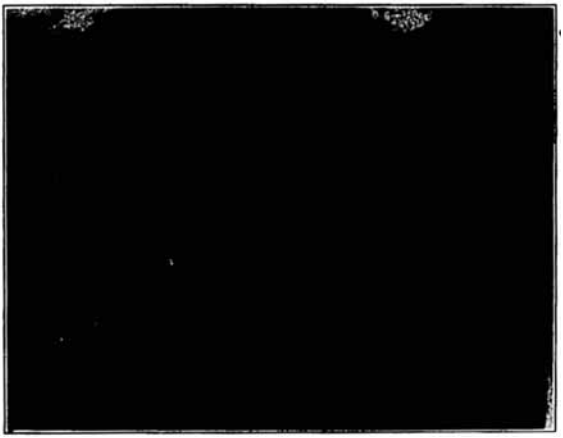

FIG. 2.—Note emphysema of supraclavicular tissues.

(Fig. 1). The upper and lower extremities were also involved. The scrotum was distended to about one and one-half times its normal size. The patient had the appearance of a nephritic.

Roentgen-ray Reports. October 14. Extensive quantities of "gas" shown in tissues of chest, neck (Fig. 2), arms down to wrist, over abdominal muscles and through the pelvis, scrotum and legs (Fig. 6).

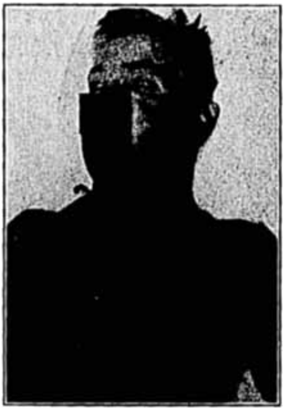

Fig. 3.—Note emphysema of neck and upper chest.

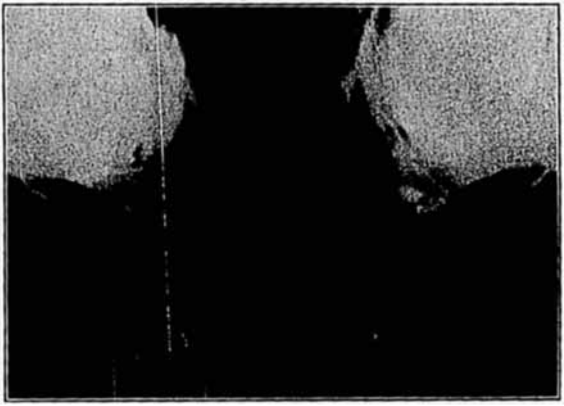

Fig. 4.—Note emphysema of neck.

October 17. Spot shadows throughout both lungs. Dilated heart. "Gas" in quantities throughout trunk, pelvis, legs and arms and wrists.

October 19. "Gas" in the legs has diminished; chest and abdomen remained the same.

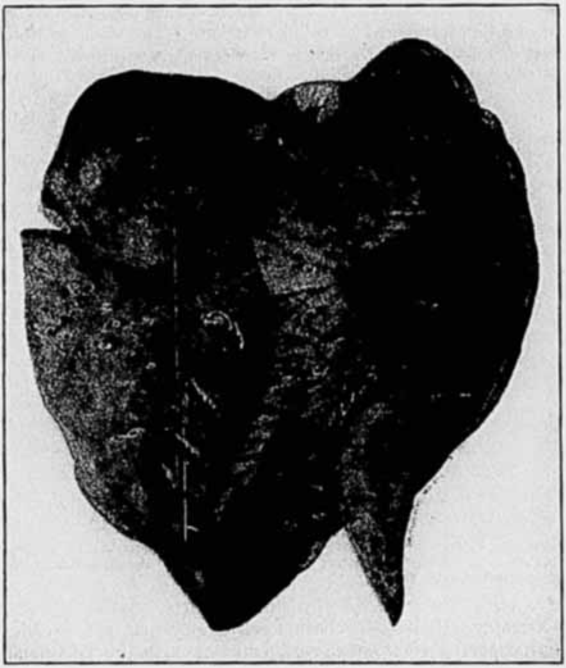

FIG. 5.—Acute ulcerative bronchiectasis; note the apparent necrotic condition of the walls.

October 14. Pneumococcus Type I reported in the blood and patient was given 100 c.c. antipneumococcus serum; this was repeated on the next day.

October 16. Patient complained of pain in the right side.

October 17. Fifty cubic centimeters of antipneumococcus serum. No difficulty in locating veins for intravenous injection and no apparent increase in the emphysema after the severe chill following the initial injection of serum. Patient showed no improvement in general condition, but emphysema diminished, particularly about the face.

October 20. Patient died.

Blood culture: Pneumococcus Type I. Leukocytes: October 3, 4000; October 10, 3400; October 15, 10,000. Anaërobic and aërobic cultures of "gas."

The skin was punctured under aseptic precaution and sterile salt solution introduced by capillary pipette. The washings were then cultured. Negative results after seven days' incubation at 37° C.

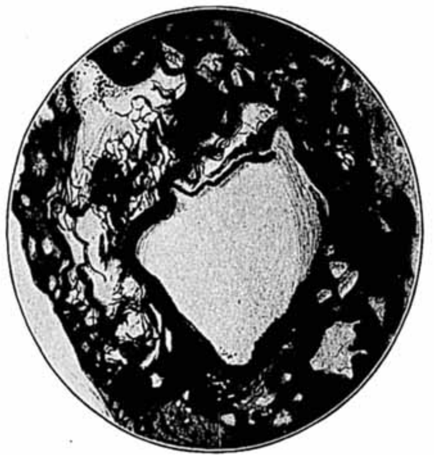

FIG. 6.—Ulcerative bronchitis; infiltration with air of peribronchial tissues. Note the torn, necrotic condition of the bronchial wall and the distention of the peribronchial areolar tissues.

Necropsy (18-196). Subcutaneous emphysema; left eyelids, left temporal region, cheeks and entire neck, entire chest and abdomen to anterior scapular line without involving gluteal region, left side of scrotum, left lower extremity to knee, right lower extremity to thigh. Subfascial and fascial emphysema of neck, axilla, chest and abdomen. Slight icterus. Emphysema of mediastinum. Edema of areolar tissue of mediastinum. Acute fibrinous pleuritis, right. Early lobular bronchopneumonia in left upper lobe. Confluent bronchopneumonia in left lower lobe. Compensatory inflation on anterior edge of entire lung, left. Bronchopneumonia, upper and lower lobe; compensatory inflation, anterior edge, right lung. Purulent bronchitis with acute ulcerative bronchiectasis, more marked in left lung. Acute fibrinopurulent bronchitis. Cloudy swelling and slight right-sided dilatation of heart. Acute follicular splenitis. Congestion and edema of adrenals. Acute parenchymatous nephritis. Cloudy swelling and terminal congestion of liver. Cloudy swelling and congestion of the pancreas. Lymphoid hyperplasia and congestion of intestines. Edema of pia-arachnoid; congestion of brain.

Microscopic Findings: Lung: fibrinopurulent bronchopneumonia; areas of hemorrhage; marked edema; acute ulcerative bronchiectasis; dilatation of peribronchial spaces; areas of necrosis; acute emphysema. Heart: congestion; cloudy swelling; conglutination and hyaline thrombi; acute fibrinous pericarditis. Spleen: acute diffuse splenitis; massive hemorrhages; partial necrosis of reticulum. Adrenal: lipoid exhaustion; congestion. Kidneys: acute glomerulotubular nephritis. Liver: congestion; cloudy swelling; focal toxic necrosis. Brain: cloudy swelling of ganglion cells; congestion.

Case II.—Private M. Admitted February 18, 1919. Previous history: measles, pertussis, scarlet fever. Onset: two days before admission he complained of cough, chilly sensation, fever. On admission: a young, well-nourished man, apparently not very sick, complaining of headache, pain in the extremities and cough. Examination: no definite lung finding. A diagnosis of influenza was then made on the basis of general appearance. Patient showed no change until three days later, when many rales were noted throughout the chest.

February 22. Left lower dulness, distant bronchial breathing and fine crepitant rales; right lower dulness and rales.

February 24. Complained of intense pain in the right chest. Examination showed dulness, bronchial breathing, rales; no evidence of friction rub. On the left side the physical findings were unchanged, but notation was made of diminished breath sounds on left side.

February 25. Condition poor, pulse could not be obtained, cyanosis intense; hyperresonance over left chest anteriorly; in the left midaxillary line: dulness, breath sounds present. The heart borders could not be outlined on percusion; no visible apex impulse. On auscultation over the pulmonic area a loud crackling sound was heard as the stethoscope was placed on the skin. On palpating this area was distinct; fine crepitations in the subcutaneous tissue were obtained. This area extended from the second intercostal space to the nipple proper. The breath sounds were diminished; the heart tones were not audible. The clinical picture at once suggested a pneumothorax with subcutaneous emphysema. 4 P.M.: The emphysema had spread to the clavicle on the left side and could be felt over the sternum. It also extended to a slight degree below the left nipple at 6 P.M.: subcutaneous emphysema had extended laterally on the left side and reached the midaxillary line. The patient was very cyanotic. The upper extremities were cold and covered with a clammy perspiration. Patient complained of pain and numbness of feet; this complaint commenced at 12 P.M. The feet were cold; there was no pulsation in the dorsalis pedic artery, but the femoral pulse and sounds could be obtained. The abdomen was tender and slightly distended. 7 P.M.: emphysema had not spread; pulse could not be obtained. 8. P.M.: patient died.

Roentgen-ray Reports. 1. Spot shadow, right lower, February 18. 2. "Gas" in the subcutaneous tissue of left chest. No evidence of pneumothorax.

Laboratory Reports. Sputum: Streptococcus hemolyticus. Blood Culture: Negative. Leukocytes: February 24, 6900.

Necropsy (19–364). Subcutaneous emphysema of neck, left upper chest, upper part of left abdomen. Emphysema of anterior mediastinum. Left pneumothorax. Zenker's hyaline degeneration of abdominal muscles. Bilateral acute fibrinous pleuritis. Confluent bronchopneumonia of left basal lobe. Patchy bronchopneumonia of left upper lobe. Inflation of lung borders. Confluent bronchopneumonia of right upper and lower lobes; inflation of borders. Purulent bronchitis. Cloudy swelling and dilatation of right heart. Acute follicular splenitis. Congestion of adrenals. Congestion of kidneys; acute parenchymatous nephritis. Acute tonsillitis. Cloudy swelling of liver. Cloudy swelling of pancreas. Slight congestion of intestines. Slight congestion and edema of pia-arachnoid. Petechial hemorrhages of brain.

Microscopic Examination. Lung: fibrinocatarrhal bronchopneumonia. Ulcerative bronchitis. Marked congestion. Heart: slight congestion; cloudy swelling. Spleen: acute follicular splenitis. Adrenal: lipoid exhaustion; edema; congestion. Kidneys; extreme congestion; acute tubular nephritis. Liver: cloudy swelling; biliary pigmentation. Brain: focal hemorrhage; cloudy swelling of ganglion cells.

CASE III.—Private B. Admitted October 3, complaining of sorethroat.

October 9. Many rales left base posteriorly; temperature, 104.4; pulse, 112; respiration, 48.

October 13. Patient was reported as a case of subcutaneous emphysema and notations on the brief showed it had developed on the left side, upper part of the chest, on the evening of October 13.

October 14. Examination showed a very robust young man, cyanotic and dyspneic, very uncomfortable and toxic. The emphysema involved the face on both sides and the patient had the appearance of a case of mumps; palpation revealed definite crepitation. The eyelids were not involved except the right, which was but slightly swollen. Both sides of the neck were distended (Fig. 3). "Gas" was also present in the subcutaneous tissue of the chest anteriorly and posteriorly. The abdomen, anteriorly, appeared distended; the skin was "ballooned" up from the underlying tissues as if a layer of air were interposed; on deep palpation crepitation was obtained. Both upper and lower extremities were involved; the scrotum was distended. There was no change in the clinical picture until October 17, when the patient began to complain of pain in the region of the larynx. Laryngoscopic examination on October 18 showed congestion of larynx, with some purulent secretion; cords normal.

October 19 to 24. The emphysema gradually diminished and the patient appeared to be improving.

October 25. Complained of pain on swallowing and dyspnea.

October 26. Exploratory aspiration of the right chest; needle used for local anesthesia was used as aspirator, no fluid but air returned. A larger needle was introduced and returned a large amount of air without blood or fluid. Comfortable during and immediately after aspiration. Thirty minutes later intense dyspnea, rapid pulse, dilated pupil.

October 27 and 28. Emphysema diminishing, condition poor; influenza immune serum intravenously.

October 29. Patient died.

Roentgen-ray Reports. October 14. Roentgen ray; extensive quantities of air throughout subcutaneous tissues of neck and chest, apparently following fascial planes over the abdomen, pelvis and gluteal region, right arm to elbow and left arm to elbow, left leg below knee and right leg below knee. Extensive spot shadow throughout lungs.

October 16. Gas showed more extensively throughout body (Fig. 4), more particularly in arms and legs.

October 17. Spot shadows throughout both lungs. Dilated left heart. Gas over the trunk, and arms to wrist, and legs half-way below knee.

October 18. The gas throughout tissues over trunk somewhat less prominent, but well marked in right arm.

October 19. Extension of gas in right hand. Chest and abdominal condition improved.

October 21. Gas in tissues over chest, neck, abdomen, thighs and arms appeared to be less than on previous day.

Laboratory Reports. White counts: October 16, 16,200; October 18, 17,500; October 25, 13,900. Sputum: Non-hemolytic streptococcus; staphylococcus; pneumococcus (type not determined). Urine: Many hyaline and granular casts. Specific gravity, 1028.

October 14. All cultures, aërobic and anaërobic, negative.

Necropsy (18–216). Subcutaneous emphysema of entire chest and abdomen. Prelaryngeal abscess. Acute bilateral fibrinous pleuritis. Confluent bronchopneumonia of both lungs. Compensatory inflation of anterior edges of both lungs. Acute purulent bronchitis and bronchiectasis. Acute diffuse splenitis. Cloudy swelling and congestion of kidneys. Cloudy swelling and terminal congestion of liver. Acute adenitis of mesenteric lymph nodes. Acute myositis and hyaline degeneration in rectus muscles. Slight edema of pia-arachnoid.

Microscopic Examination. Lungs: Fibrinocatarrhal bronchopneumonia; ulcerative bronchitis; areas of hemorrhages; acute emphysema. Heart: congestion and cloudy swelling. Peribronchial lymph nodes: marked congestion; edema; sinus catarrh. Adrenal: lipoid exhaustion; congestion and edema; hyaline thrombosis. Kidneys: congestion; cloudy swelling. Liver: hydrops; congestion; hyaline thrombosis; slight biliary pigmentation.

CASE IV.—Private P. Admitted October 10, 1918. Previous history negative. Was sick three or four days before admission to hospital, complaining of severe backache, pain in the chest, vomiting, slight coryza; throat injected, a few scattered rales in both bases. The patient showed no clinical change until ten days later, when he developed a rigid neck and positive Kernig's sign. A spinal puncture was performed but proved to be negative, microscopically and bacteriologically.

October 21. Patient had marked difficulty in breathing and swallowing; examination revealed an acute laryngitis, acute inflammatory edema of the glottis, epiglottic folds and arytenoids. Aryepiglottic folds and arytenoids were markedly congested, interfering with respiration. High tracheotomy was performed under local anesthesia.

October 24. One of us was called in to see the case at this time and distinct crepitation was present in the tissue of supraclavicular spaces and neck. On observation during the day it was noted that the gas remained localized in neck region but increased in quantity.

October 25. Patient died.

Roentgen-ray Reports. October 12. Spot shadows in both lower lobes. Throat culture: Staphylococcus predominating. Leukocytes: October 12, 5800. Sputum: Staphylococcus; pneumococcus.

Necropsy (18–209). Subcutaneous emphysema of anterior neck. Operative wound of neck (tracheotomy). Cervical abscess. Bilateral fibrinopurulent pleuritis. Bilateral lobular bronchopneumonia of all lobes. Acute diffuse splenitis. Massive hemorrhage of right adrenal. Acute parenchymatous nephritis; congestion of kidney. Cloudy swelling of liver. Congestion and edema of pia-arachnoid. Lymphoid hyperplasia of intestines. Acute suppurative otitis media, right. Acute mastoiditis.

Microscopic Examination. Lungs: acute fibrinocatarrhal bronchopneumonia; edema; marked congestion; ulcerative bronchitis; acute inflation. Heart: congestion; cloudy swelling; peribronchial lymph nodes: congestion and edema; sinus catarrh. Spleen: acute diffuse splenitis. Adrenal: lipoid exhaustion; congestion. Liver: congestion; cloudy swelling. Kidneys: acute glomerulotubular nephritis. Brain: cloudy swelling of ganglion cells; congestion; minute hemorrhages.

Case V.—Lieut. S. Admitted to Base Hospital October 3, 1918. Previous history negative.

October 1. Onset, with general malaise and chilliness. On admission a robust young man, rather dyspneic, coughed persistently. Intense cyanosis of face; dulness of right lower lobe; bronchial breathing.

October 7. Extensive involvement of right lower and middle lobes, with dulness, bronchial breathing, crepitant and subcrepitant rales. Many small areas of dulness with many rales in the left side. Cyanosis and dyspnea very pronounced.

October 10. Distant crepitation over the entire right side of the chest and neck. The face was not involved.

October 11. Patient died.

Roentgen-ray Reports. October 8, 1918: shadows involving both lower lobes. Sputum bacteriology: streptococcus, pneumococcus, staphylococcus, M. catarrhalis.

Leukocytes: October 8, 1918, 6500.

Case VI.—Private G. Admitted October 3, 1918. Previous history: exanthemata, including smallpox, when a child; fracture of ribs several years ago. Onset sudden, cough, pain in chest and fever. On admission: a robust young man, appearing not very sick, with cyanosis of face; throat purple and injected; no definite evidence of consolidation, many rales. Diagnosticated severe influenza.

October 5. Consolidation lower left.

October 8. Subcutaneous emphysema in uper part of chest, neck, face. Patient delirious; petechial eruption on skin.

October 9 to 12. No change; clear mentally.

October 18. Emphysema of subcutaneous tissue diminished; absent on chest.

October 21. Lungs clear; breath sounds high pitched; no rales.

November 1. Discharged from hospital as recovered.

Roentgen-ray Reports. October 14. Extensive quantities of gas in the tissues over neck, shoulders, abdominal muscles. Spot shadows throughout right lung. Shadow in lower left. Gas over gluteal region, thigh, legs and forearms.

October 16. Gas more extensive than on previous report.

October 18. Gas less marked in tissue than on previous report. Leukocytes: 7500.

Case VII.—Private R. Admitted October 3, 1918, with diagnosis of influenza.

October 9. Many rales over right lower lung.

October 14. Developed bilateral lobular pneumonia.

October 15. Patient very weak, coughed persistently, sputum bloody.

October 16. Cyanosis of face. Mental condition sluggish.

October 18. Patient appeared very toxic and was delirious. Spinal puncture performed and 20 c.c. of clear fluid removed; this showed no increase in cells and no bacteria; globulin negative.

October 20. Condition serious; patient dyspneic and cyanotic. Subcutaneous emphysema developed, involving the face, neck, chest and upper and lower extremities bilaterally.

October 21. Patient's condition serious. The emphysema in the lower extremities diminished.

October 22. Subcutaneous emphysema had extended to waist line. Lower extremities free.

October 23. Subcutaneous emphysema increasing.

October 25. Intravenous injection of immune serum.

October 26. Weak; complains of pain in chest.

October 29. Emphysema disappearing. Gradual improvement. Discharged from hospital November 22, 1918, as cured.

Roentgen-ray Reports. October 15. Shadow in lower right.

October 20. Extensive spot shadows in lower right with same on left. Gas showing throughout muscles of trunk with some in the thighs, also in forearms.

October 21. Gas in tissues of neck, chest, abdomen and arms.

October 23. Gas extending.

In addition to the above one of us has observed a similar condition complicating measles. The emphysema was localized in the chest wall; the patient recovered. In another instance generalized subcutaneous emphysema developed postoperatively in a patient who had a pyopneumothorax. In this case the emphysema developed one day after operation; the patient was aspirated repeatedly prior to operation but never presented evidence of subcutaneous emphysema. When the emphysema did develop it was rapid and generalized, involving face, neck, chest, abdomen and extremities. This patient died.

Clinical Analysis. An analysis of these cases from the clinical aspect reveals certain constant features.

The onset of the subcutaneous emphysema was apparently sudden, but after its appearance the "gas" spread relatively slowly, attaining its greatest extent in twenty-four to forty-eight hours.

The time of appearance of the emphysema, relative to the stage of influenza pneumonia, was not definite, but it is safe to say that in the majority of the cases the "gas" developed when the pneumonic process was well advanced. Table I shows this relationship.

TABLE I.—TIME OF DEVELOPMENT OF EMPHYSEMA.

Case No.	Influenza.	Pneumonia.	Emphysema.	Death or recovery.
I . .	October 2	October 9	October 9	October 19
II . .	February 18	February 22	February 25	February 25
III . .	October 3	October 5 or 9	October 14	October 29
IV . .	October 10	October 10	October 24	October 25
V . .	October 3	October 5 or 7	October 10	October 11
VI . .	October 3	October 5	October 8	Recovered.
VII . .	October 3	October 14	October 20	Recovered.

It is noteworthy that the paroxsymal cough, to which has been ascribed an important place in the possible etiology of the subcutaneous emphysema, occurs very early in the picture of influenza pneumonia, and that when the pneumonic process is well established cough is not common. It might be assumed that with a generalized pulmonary emphysema, or with a destructive softening of the lung parenchyma (Torrey and Grosh[7]) and an associated severe cough, rupture of alveoli could easily occur and be the source of emphysema. The striking contrast, however, of the late appearance of the emphysema and the absence of cough during that period suggest that the cough probably plays a minor role.

Symptoms associated with the development of the subcutaneous emphysema were, according to our experience, practically negative. The patient was generally unaware of the complication until it was pointed out to him. The temperature, pulse and respiration showed no change. Torrey and Grosh,[7] however, state that "when in apparently respiratory extremis a patient would complain of pains, substernal and in the jugular fossæ, and crepitation would be noted in the subcutaneous tissues at the root of the neck, an immediate marked subjective relief was apparent, rapidly followed by a noticeable improvement in the respiratory excursion of the chest and the most striking decrease in cyanosis and jugular distention." Measurements of the respiratory excursion showing "that there was apparent an increase in the chest circumference during the illness and an abnormally small respiratory excursion, with a striking increase immediately as the subcutaneous emphysema gave vent to the air contained in the mediastinum."

We observed precordial pain in but one case, but pleuritic pain was common. Cyanosis was not relieved to any marked degree nor was the respiratory rate diminished as shown by our clinical record. Table II shows that the temperature, pulse and respiratory rate, before and after the appearance of the subcutaneous emphysema, were practically unchanged.

LOCATION OF SUBCUTANEOUS EMPHYSEMA. The subcutaneous emphysema has developed most frequently in the neck region. In one case it was first noticed in the region of the second left intercostal space. The supraclavicular spaces, neck, chest, face, extremities, abdomen and scrotum, have been involved. It is easily diagnosticated by obtaining a fine crepitation on palpation, and on auscultation the stethoscope produces a loud crackling sound as it comes in contact with the skin.

ABSENCE OF SIGNS OF INFLAMMATION. There is no apparent change in the leukocytes with the development of the emphysema. Likewise there is no local skin eruption or inflammation. The physical condition seems but slightly affected by the development

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[7] AM. JOUR. MED. SC., 1919, clvii, 170.

TABLE II.—TEMPERATURE, PULSE, AND RESPIRATION BEFORE AND AFTER APPEARANCE OF EMPHYSEMA.

Date.	Before emphysema developed.			Date.	After emphysema developed.		
	Temp.	Pulse.	Resp.		Temp.	Pulse.	Resp.
Case I:							
Oct. 7	105.0	112	28	Oct. 9	103.0	110	26
	100.0	92	26		101.0	90	30
Oct. 8	104.0	116	24	Oct. 10	101.8	84	24
	101.4	100	26		101.8	100	26
				Oct. 15	101.8	110	30
					100.2	84	40
Feb. 22	100.0	92	22				
	102.2	92	22				
Case II:							
Feb. 23	102.8	90	24				
	102.0	110	28				
	102.6	108	24				
	102.6	92	24				
	103.0	112	26				
Feb. 24	103.6	90	24	Feb. 25	103.2	120?	36
	104.0	108	46		104.0	120?	36
	104.4	136	42		100.0	?	40
	103.8	120	36		102.0	?	48
	102.0	120	36				
Case III:							
Oct. 10	104.4	102	48	Oct. 13	100.0	116	44
Oct. 11	103.6	104	48		101.0	118	40
	103.8	112	36	Oct. 14	98.0	70	26
Oct. 12	101.0	98	36		100.0	110	30
	101.6	140	40	Oct. 15	100.0	100	22
					100.0	100	22
				Oct. 16	101.0	120	28
				Oct. 17	98.0	118	36
					101.0	120	30
Case V.							
Oct. 8	103.0	98	36	Oct. 10	103.0	122	54
	102.0	140	38		102.0	120	56
	104.0	120	28				
Oct. 9	103.6	120	46				
	103.0	140	60				
Case VI:							
Oct. 7	101.0	126	30	Oct. 8	101.0	96	30
	102.0	100	46		102.0	120	32
				Oct. 9	100.0	98	42
					102.6	120	42
				Oct. 10	101.2	96	30
Case VII:							
Oct. 18	99.2	106	36	Oct. 20	103.2	144	48
	99.8	100	32		102.2	120	32
	101.2	110	44	Oct. 21	100.4	130	40
Oct. 19	99.0	120	46		102.4	128	52
	99.4	120	52		102.2	120	54
	98.8	140	52		102.8	120	44
	100.0	126	40	Oct. 22	101.0	104	44
					101.0	124	56
					101.2	112	50
					101.2	122	54

of even most extensive emphysema. In all of the patients the emphysema subsided considerably as the disease progressed and death was always due to associated conditions. Two of the cases recovered entirely.

Of the cases which developed subcutaneous emphysema it is noteworthy that in addition to the severity of the picture of pneumonia there were additional complications. Thus delirium occurred in one patient, meningismus in another and possibly a slight pneumothorax in still another. Prelaryngeal abscess and edema of the glottis were also encountered. The association of subcutaneous emphysema following tracheotomy has been reported previously,[8] but not in association with pneumonia. In Case IV the occurrence of an emphysema following tracheotomy is noteworthy, since wounds of the larynx and trachea[9] have been given as a cause of emphysema, but it seems evident that the emphysema in Case IV was not due to the laryngeal pathology.

In attempting to elicit a possible etiological factor in these cases we questioned our patients as to trauma and previous history of gas attacks. In none of these was any such history obtained.

The occurrence of only one case of pneumothorax diagnosticated clinically and the constant absence of any evidence of pneumothorax on roentgen-ray examination in all our cases of subcutaneous emphysema is emphasized.

TREATMENT. The treatment in general was directed to the pneumonia or the associated complication rather than to the emphysema. As has been stated the development of the subcutaneous emphysema was not accompanied by any symptoms. The emphysema apparently will diminish or disappear without any operative or medicinal interference.

DISCUSSION OF ANATOMICAL FINDINGS. The subcutaneous emphysema in two of the cases was considerably more marked on the left than on the right side. The significance of this will be commented on later. The external appearance of the bodies has been described in the clinical part of this paper, and it need only be added that two of the patients presented the extreme lividity of the face and the body so frequently seen in fatal influenza.

The subcutaneous tissues when incised were moderately moist and slightly bloody, and, in a general way, had the appearance of innumerable small air bubbles in the alveolar tissue. Over the chest this distended areolar tissue measured about 15 mm. in its thickest part, over the abdomen about 5 mm. Sections from this tissue presented none of the changes associated with inflammation. Coagulation necrosis of the rectus muscles was met with in two cases;

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[8] Ashley and Wright: Diseases of Children, American edition, 1893. Quoted by Alexander, M. E., and Follett, E. C.: Jour. Am. Med. Assn., 1919, lxxii, 930.

[9] Lexer, E.: General Surgery. Quoted by Alexander, M. E., and Follett, E. C.: Jour. Am. Med. Assn., 1919, lxxii, 930.

this condition was also present in non-emphysematous influenzal patients.

The mediastinum in two of the bodies resembled the subcutaneous tissues in general appearance. In two other bodies no changes were noted. In the first patient the areolar tissue throughout the mediastinum was edematous as well as emphysematous, forming a large mass over the pericardium, which, when dissected out, was found to weigh 165 grams. Otherwise the gross and minute anatomical changes in the various organs were similar to those in other influenza cases, and since a study of this disease has been published by one of us,[10] it would lead to unnecessary repetition to enumerate them here. We shall confine ourselves, therefore, to a description of the respiratory tract lesions, and these, for the sake of brevity, are discussed collectively.

Lungs. It may be stated that the lungs differed in no way from the lungs of other influenzal patients. The left lungs varied from 490 to 1040 grams in weight and the right lungs from 590 to 850 grams. The anterior edges of the lungs were in each case moderately inflated, but this was never more marked than in other patients which did not have subcutaneous emphysema. In no case were air bladders or pearl-like distended air vesicles seen, although this was not infrequently encountered in our other influenza autopsies. The surface of the lungs was carefully examined to note any point of possible rupture, but this could in no instance be demonstrated. The posterior parts of the lungs were more or less consolidated, the areas of bronchopneumonia tending to become confluent. The extent of pneumonic involvement was never more marked than in the average influenza pneumonitis. The pleuritic complications were of the type usually met with. In no instance were the lungs firmly adherent to the lateral chest wall.

The cut surface resembled that usually seen in influenza pneumonitis, that is, it was generally very bloody and very moist, with large, poorly circumscribed, consolidated areas, some of which were granular, others entirely smooth. The bronchial branches, in all but one instance, contained large quantities of thick, creamy pus. The bronchial mucosa was swollen and deeply hemorrhagic. Once the walls of several of the larger bronchial branches were definitely eroded and the lumen markedly dilated, forming acute ulcerative bronchiectases (Fig. 5). In another instance cylindrical dilatation of some of the bronchial branches was present.

The microscopic examination of the lung presented the usual hemorrhagic, fibrinocellular exudate of influenzal pneumonitis. In all but one instance there were areas of necrosis, usually surrounding a destroyed bronchiole, but occasionally seemingly independent of the bronchial tubes. Once marked thickening of the interpulmonary septa occurred. The bronchial branches in each instance presented here and there partial or complete erosion of their walls, masses of necrotic debris and fibrin occupying the site of the destroyed areas. Once the peribronchial tissue was found markedly distended, giving the appearance that air had escaped through the destroyed bronchial wall in the loose peribronchial tissue (Fig. 6). While ulcerative bronchitis was not uncommonly met with in about 200 cases of influenzal pneumonitis, which we have examined here postmortem, it appeared regularly in the four cases complicated with subcutaneous emphysema. Ulcerative bronchiectasis of the larger bronchial branches was uncommon in the epidemic here, occurring only twice outside of the case reported here.

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[10] Lucke, Baldwin, Wright, Toynbee and Kime, Edwin: Pathological Anatomy and Bacteriology of Influenza, Arch. Int. Med., August 15, 1919.

Bacteriological cultures were made during life from the subcutaneous tissues at various parts of the body, and after death from the lungs, pleural exudate, subcutaneous tissues, heart's blood, spleen, brain, and other organs. As has been stated in the clinical part of this communication, all the antemortem cultures were negative; the postmortem cultures presented in each case hemolytic streptococci, pneumococci, and influenza bacilli, either alone or in combination.

DISCUSSION OF THE MODE OF ORIGIN OF SUBCUTANEOUS EMPHYSEMA. It may be well to briefly review the various theories which have been suggested as the cause of this unusual complication:

Bacterial activity may be at once dismissed, since no local inflammatory reactions point to this condition. Torrey and Grosh speak of a destructive softening of the lung parenchyma, due probably to alteration of the lung tissue by toxin or virus. Chemical changes in the blood, permitting escape of air into the tissues is a possibility. The delayed time of the coagulation of the blood, the frequency of nose-bleed, petechial hemorrhages throughout the body, allows one to entertain the thought that chemical blood changes may occur which prevent the blood from maintaining the normal balance of its gases. We have analyzed the air obtained by puncture of the distended scrotum of one of the patients examined and found it to resemble atmospheric air.

Most of the writers who have reported cases of subcutaneous emphysema complicating influenza maintain that this condition is purely mechanical in origin. Thus, Symmers says, "In two cases rupture of the distended air vesicles has taken place probably near the apex of the lung. In another rupture occurred in the left pleural cavity, with the production of pneumothorax. In none of these cases was it possible to demonstrate at necropsy the actual point of rupture." Berkley and Coffen report 11 cases, 2 of which were associated with spontaneous pneumothorax. They suggest that subcutaneous emphysema may occur through the intrapleural route, providing that adhesions exist between the two layers of pleura at the point of rupture, or through the extrapleural route if the air dissects its way to the hilum of the lung. Clark and Synnott, in the report of 12 cases, conclude that the origin of the gas was from weakened alveoli of the lung under pressure.

We believe that subcutaneous emphysema chiefly arises by escape of air from ulcerated or eroded bronchi and its passage along peribronchial or perivascular channels into the mediastinum. The clue of our explanation, in at least 2 of the cases, was found in the occurrence of marked ulcerative bronchitis and subcutaneous emphysema, both located on the left side.

If we disregarded simple mechanical rupture of peripheral alveoli as a highly improbable factor in the production of subcutaneous emphysema, there remain, then, only three possible ways for pulmonary air to reach the subcutaneous tissues: (*a*) by necrotizing destruction of alveoli; (*b*) by ulcerative or erosive rupture of a bronchus; (*c*) by rupture of pulmonary tissue through adherent pleural surfaces. By necrotizing destruction, alveolar air could pass into the loose peribronchial or perivascular areolar tissue and thence into the mediastinum. By ulceration or erosion of a bronchus the air would likewise gain the mediastinum. In neither case would the air pass into the pleura, because this is a closed sac. The third way is through perforation of necrotic pulmonary and overlying pleural tissue into the pleural cavity, resulting in pneumothorax. Should the visceral and parietal layers of the pleura adhere to one another the air could penetrate both layers and dissect the areolar tissue between the parietal pleural and thoracic wall to the mediastinum. Such a case would necessitate, it seems, a considerable area of adhesion. Once the air has found a vent from the pulmonary tissues, expiratory force and elasticity of the chest wall would further drive it along the lines of least resistance, which is into the neck (the location in which the subcutaneous emphysema apparently always began). Thence, along fascial planes, the emphysema would become generalized.

Microscopic examination presented a distention of the peribronchial areolar tissue (Fig. 6), and this we consider a support of our second explanation of the escape of air from the lung.

The question would naturally arise, "Why should this condition be so uncommon when ulcerative bronchiectasis is not infrequently met with in various diseases, as, for instance, tuberculosis. But it must be pointed out that bronchiectasis rarely occurs acutely and that the reaction of the tissues surrounding the bronchi would probably form a wall impenetrable to the air. It may be assumed, then, that only in rapidly occurring bronchiectasis, without sufficient tissue reaction, is such a condition possible, and that they arise but rarely. It would seem, likewise, that a fair-sized bronchial branch is necessary to allow sufficient air to escape for the production of emphysema. This would appear to us more likely as the cause than mechanical rupture of peripheral alveoli, for not only are hugely distended and sometimes ruptured alveoli frequently encountered in both acute and chronic pulmonary emphysema, but the rupture of a few air sacs would not likely lead to a widespread air infiltration. Further, rupture of peripheral alveoli could only produce emphysema, provided that firm adhesions were present between the pleural layers at the point of rupture. Pneumothorax would result otherwise and in none of the cases reported has this occurred frequently. In the one instance where pneumothorax was present in our series, it was not at all marked, since only a few small air bubbles escaped upon puncturing chest wall through a pocket filled with water.

It is not at all unlikely that erosion or rupture in a bronchial tube might be overlooked, and this, we feel, rather strengthens than weakens our argument. The absence of mediastinal emphysema in two of our cases may be readily explained by the gradual absorption of air which occurred in every one of these cases, for, as has been stated, a marked reduction in the extent of the subcutaneous emphysema took place in every instance. This probably is due to the gradual closing up of the point of eruption because of cellular reaction.

Conclusions. 1. Subcutaneous emphysema is an exceedingly rare complication of influenza.

2. Previous explanations of the condition seem inadequate and unsatisfactory.

In two of our postmortem cases we encountered a combination of more pronounced left-sided subcutaneous emphysema with left-sided ulcerative bronchitis, and on microscopic examination the peribronchial tissue was found greatly distended. We believe the bronchial air to have escaped into the loose peribronchial tissues, and by dissecting along lines of least resistance to have gained entrance to the mediastinum and thence, along fascial planes, to the subcutaneous tissues. In this way the closed pleural sacs are not involved, and this accounts for the infrequency of pneumothorax in this condition.

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GROSS PATHOLOGY OF INFLUENZAL PNEUMONIA IN FRANCE.

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The material for this paper was collected from 106 autopsies on cases of pneumonia dying at a base hospital in France (Commercy,